pletely both pyramidal tracts in the anterior pyramids, or in the cerebral peduncles—in the latter case cutting also the so-called pontile tracts of the crusta—after he had anesthetized the motor cortex, and he then obtained contraction of the muscles by cortical irritation with the faradic current. He believes he has demonstrated the existence of motor "extrapyramidal" tracts and of their decussation in the medulla oblongata, because he prevented all reflex action by the anesthetization of the cortex, and because the muscular contractions could not have been caused by way of the pyramidal or pontile tracts. He believes also that the "extrapyramidal" tracts are for associated movements; for example, he obtained extension of the foreleg on the side opposite to, and of the fore and hind legs, on the same side as the irritated anesthetic cortical area, after he had cut both pyramidal tracts. This he regards as an associated movement.

Prus found also that when he cut one-half of the midbrain just behind the posterior quadrigeminal bodies, and then irritated the cerebral cortex of the same side, he was able to obtain bilateral epileptic attacks. When he further cut one-half of the lower portion of the medulla oblongata, or of the spinal cord, on the side opposite to the injured half of the midbrain, he was still able to obtain bilateral epileptic convulsions from cortical irritation. These experiments showed that the epileptic attacks became general by means of the gray matter of the midbrain, medulla oblongata and spinal cord.

There is no special epileptic center, and there is no essential difference between cortical and "genuine" epilepsy. SPILLER.

66. UNTERSUCHUNGEN UBER DIE FEINERE STRUCTUR DER NERVENZELLEN UND IHRER FORTSÄTZE (Investigations of the Finer Structure of the Nerve Cell and its Processes). Vladislav Ruzicka (Archiv. f. Mikroskopische Anatomie und Entwickelungsgeschichte, 53, 1898, p. 485.)

The author presents the following conclusions:

(1.) The Nissl bodies are not preformed in the living nerve cell. They are artefacts, not produced, as maintained by Held, by acid fixatives, but by the process of decolorization.

(2.) The anterior horn motor cells of the ox and dog do not possess throughout, a parallel striped protoplasmic structure. This arrangement, when it exists, cannot be held to be characteristic for a motor-acting cell.

(3.) The nerve cells of the spinal cord may be united to one another by anastomoses.

(4.) The nerve cells of the spinal cord send out fine processes from their surfaces which pass into the surrounding tissues.

(5.) The larger dendrites as well as the finer processes of the nerve cell probably act in some anabolic capacity. JELLIFFE.

67. BEITRÄGE ZUR HISTOPATHOGENESE DER TABISCHEN HINTERSTRANGDEGENERATION (Contributions to the Histopathogeny of the Tabetic Degeneration of the Posterior Columns.) Karl Schaffer (Deutsche Zeitschrift für Nervenheilkunde, 13, 1898, p. 287).

Schaffer says that the cases of incipient tabes, especially the cases of general paralysis with involvement of the posterior columns, show a degeneration of areas exactly corresponding to those which have been observed in the fetus, and he believes that the tabetic degeneration is confined in its early stages to the embryonal fiber systems of Flechsig and Trepinsky. The study of degeneration of isolated posterior roots shows that a single posterior root contains fibers belonging to different embryonal systems, and that the embryonal systems are composed of posterior root fibers. As the degeneration of incipient tabes corresponds with embryonal zones, it follows that in tabes the posterior roots do not degenerate in toto, but that certain fibers of each affected root degenerate, and that these affected fibers correspond to the fetal fiber systems.

Schaffer believes that the ventral fields of the posterior columns contain exogenous as well as endogenous fibers, and that the same is probably true of Gombault and Philippe's triangular zone. Tabes is a systemic disease of the posterior columns, and this posterior column affection is secondary to degeneration of the posterior roots. The degeneration of tabes is systemic and segmental; i. e., it follows the embryonic divisions of the posterior columns, and is in the intramedullary distribution of each altered root.

Schaffer has shown elsewhere that the spinal ganglion cells are not altered in tabes, even when they are studied by the method of Nissl, and this fact he regards as a proof that the primary lesion of tabes is not in the cells of the spinal ganglia. The degeneration of the posterior roots in tabes may not always be elective, but a root may degenerate in toto, and most frequently both forms of degeneration are found in the same case. Infiltrative and vascular lesions of the posterior roots, producing nutritive and circulatory disturbances, play an important role in the causation of tabes; but a primary degeneration of the posterior roots may also occur. The intraspinal portion of the posterior root is more vulnerable than the extraspinal, and the degeneration begins in the former portion. The point of contraction in the posterior root (Obersteiner and Redlich's *locus minoris resistentiae*) is not always the part surrounded by the pia. It does not follow that a posterior root degenerates in toto, because it has been injured at a certain point. Schaffer has shown that the different tracts of the spinal cord degenerate at different periods, when the cord has been cut transversely; and it is probable that an entire posterior root may be exposed to injurious agents, and yet only certain fibers belonging to definite tracts may degenerate, or at least, degenerate within a certain time. In this way the tabetic degeneration may conform to the embryonal systems of the posterior columns, even though the whole of the posterior root may be exposed to injury. SPILLER.

68. BEITRAG ZUR KENTNISS DER „SECUNDÄR MALIGNEN NEUROME.“ (Contribution to the Knowledge of Secondary Malignant Neuroma). Habermann, (Münchener Medicinische Wochenschrift, 23 and 24, 1898).

Multiple neuromata seem to bear a close relationship to multiple fibromata of the skin. Their development produces the clinical picture of a rare disease, "neurofibromatosis" or "elephantiasis nervosum," which is apparently always congenital in origin. These neurofibromata seem to have a special tendency to undergo sarcomatous degeneration, though they may exist for years without giving any symptoms before this begins. A distinction is to be drawn between secondary malignant neuromata, and primary sarcomata of nerves.

In the first, hereditary predisposition is usually made evident by the presence of multiple fibromata, and often of some defect or deformity. In primary sarcoma these elements are wanting. Primary sarcoma soon breaks through its capsule, and forms metastases. Secondary malignant neuroma grows rapidly, but generally remains enclosed in its capsule, and has little tendency to form metastases. The rarity of